DISSERTATION ON

"A PROSPECTIVE STUDY OF CSF RHINORRHOEA AND OUTCOMES OF ENDOSCOPIC CLOSURE"

Submitted in partial fulfillment of the requirements for

M.S.DEGREE BRANCH -IV OTORHINOLARYNGOLOGY

of

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERISTY,

UPGRADED INSTITUTE OF OTORHINOLARYNGOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

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DECLARATION

I solemnly declare that the dissertation **" A PROSPECTIVE STUDY OF CSF RHINORRHOEA AND OUTCOMES OF ENDOSCOPIC CLOSURE"** is done by me at the Madras Medical College and Government General Hospital, Chennai during 2006-2008 under the guidance and supervision of **Prof. S. KULASEKARAN, M.S., D.L.O.**

This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of regulation for the award of M.S. DEGREE IN OTORHINOLARYNGOLOGY (BRANCH–IV).

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I am extremely thankful to my family members for their continuous support. Above all I thank God Almighty for His immense blessings.

CERTIFICATE

This is to certify that this dissertation entitled **" A PROSPECTIVE STUDY OF CSF RHINORRHOEA AND OUTCOMES OF ENDOSCOPIC CLOSURE"** submitted by **Dr. M.NALLASIVAM,** appearing for M.S. E.N.T.. Branch IV Degree examination in March 2009 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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CONTENTS

INTRODUCTION

Cerebrospinal fluid (CSF) rhinorrhoea is the leakage of cerebrospinal fluid from the anterior cranial skull base subarachnoid space into the nasal cavity due to a defect in the duramater, bone and mucosa.

CSF rhinorrhoea involves breach of large number of anatomical structure, viz. duramater, arachnoid, the skull base, and the mucosa of the nasal cavities and the paranasal sinuses.

The main surgical approaches for the surgical repair of CSF leaks are intracranial and extracranial.

Endoscopic guided approach for surgical repair of CSF rhinorrhoea offer the benefit of both panoramic and detailed image of the site of surgery. Owing to the specific direction of view of the endoscopic lens system, it is possible to inspect the circumference of the operating field at 360 degree, by rotating the telescope around it longitudinal axis. These technical features enables direct endonasal access to the anatomical structure at the skull base without the need for cutaneous incisions or cutting through bony segment and without dislodgement of bony structures.

AIMS OF THE STUDY

The present study of CSF rhinorrhoea is undertaken

- To analyse the incidence and distribution of cases according to age , sex , aetiology among the patients who require intense surgical therapy.
- To describe the various available investigatory tools for identifying the leaks in CSF rhinorrhoea.
- To describe the definitive site of the CSF leak.
- To describe the various techniques of CSF leak closure using endoscope.
- To analyse the outcome of endoscopic closure of the CSF leaks.

REVIEW OF THE LITERATURE

Galen first described CSF rhinorrhoea in the second century AD. He postulated that CSF was released into the nose by way of the pituitary and ethmoid regions.

1826 - Charles Miller published the first case of CSF rhinorrhoea in a hydrocephalic child with an intermittent discharge of nasal fluid. Autopsy revealed communications between the nasal and cranial cavities.

1899 - St. Clair Thompson reported the first series of patients with spontaneous CSF leaks and coined the term rhinorrhoea. He also differentiated between cerebrospinal rhinorrhoea and nasal rhinorrhoea. He did not recommend surgical intervention.

1926- Walter Dandy's 1st intracranial repair bifrontal craniotomy was the procedure of choice till 1940, it was associated with high morbidity and anosmia, with recurrence rate of 27%, and success rate of 12 to 20%

1948 - Gusta Dohlman [1] was first to describe the extracranial approach. He used a nasoorbital incision, through external ethmoidectomy using nasal turbinate and septum.

1952 - Oscar Hirsch[2] transnasal approach- he closed two sphenoid CSF leaks for acromegalic patients.

1964- Vrabec and Hallberg [3] – Repair of CSF leak in cribriform plate using intranasal approach where they did simultaneous SMR for adequate visualization and defect closed by advancement flap from the turbinate.

Lehrer and Deutsch [4] reported additional two cases using operating microscope. All the above used head light and operating microscope.

1981- Endoscopic closure of minor CSF leaks that occurred during ethmoidectomy was reported by Wigand [5] and Stankiewicz [6]

Wigand used fibrin glue to close the leak.

Papay et al. [7] reported the use of endoscopic telescope in trans septal and trans sphenoidal surgery to localize sphenoid CSF leakage

Messerklinger [8], Reck and Wissen-Siegert [9] described the combination of intrathecal fluorescein and nasal endoscopes to diagnose anterior cranial fossa CSF leaks.

Mattox and Kennedy[10] reported many patients, with CSF leaks.

Dodson et al. presented a case series successfully using endoscopes for closure.

DESCRIPTIVE ANATOMY OF SKULL BASE

EMBRYOLOGY

The anterior skull base is formed by ethmoid, frontal, and sphenoid bones. Ethmoid bone ossifies in the cartilaginous nasal capsule from three centres, one for each labyrinth and one for the perpendicular plate. Centre for the labyrinth is present from 4^{th} or 5^{th} intrauterine month, partially ossified at birth. The perpendicular plate and crista galli develop form one centre, during the 1^{st} year after birth and fuse with the labyrinth at the beginning of the second year. Both this centre and those for the labyrinth contribute to the cribriform plate.

Ethmoidal turbinates[11] arise from ridges in the lateral nasal wall. By 9^{th} to 10^{th} week six major furrows develop and reduced by fusion to three to four, separated by ridges. First ethmoid primary furrow regresses. The descending part of the first primary furrow becomes ethmoidal infundibulum. Its superior ascending part becomes the frontal recess[13] ,continuing pneumatisation of the frontal recess into the frontal bone finally results in the formation of frontal sinus.Additional furrows and corresponding ridges between them evolve into anterior ethmoidal and infundibular cells.

The Frontal bone ossifies from two centres appearing in the eighth week of intrauterine week. At birth the bone is composed of two halves, separated by frontal or metopic suture, which begin to fuse from the second year usually complete by the eighth year.

Sphenoid[14] bone is divided into two parts. Pre sphenoidal portion anterior to the tuberculum sellar, continues with the lesser wing made of six separate ossification centres and the post sphenoidal part composed of sella turcica and dorsum sellae associated with the greater wings and pterygoid process derived from eight centres. Both fuse around the eighth month of intrauterine life. At birth it consists of three pieces. Central portion, body and lesser wing. Two lateral parts each consisting of the greater wing and pterygoid process which begin to fuse at one year after birth.

ETHMOID BONE AND ROOF[15]

The ethmoid bone is a paired bony scaffold which is held together by a horizontal plate which is the lamina cribrosa (Cribriform plate). Between the two lamina cribrosae, there is an anteriorly and superiorly pointing spur, the Crista galli. Across from the crista medially along the entire length between the two lamina cribrosa and at right angles to them inferiorly is the perpendicular plate.

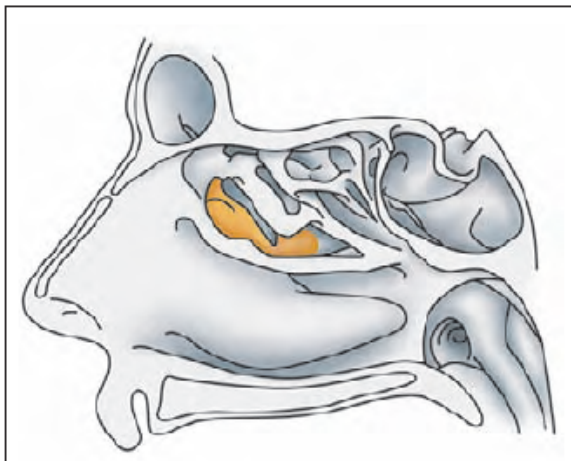
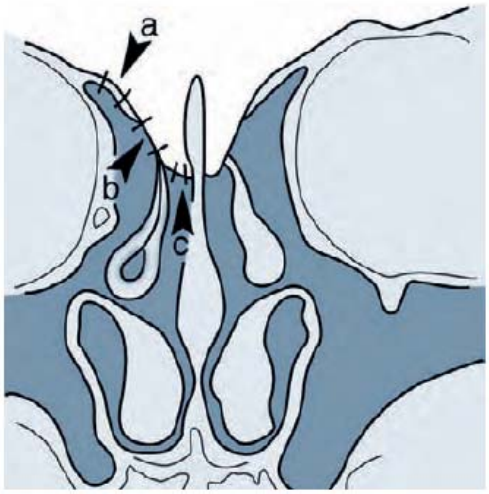


Figure showing the anterior skull base

(figure. 1)



Coronal cut showing **a** - roof of the ethmoid sinus

b - lateral lamella, **c** - cribriform plate (figure. 2)

Ethmoidal labyrinth, that is attached to the lamina cribrosa makes up the bulk of the ethmoid. Laterally the lamina papyraceae form a thin bony divider from the orbit. Medially, towards the nasal cavity, the ethmoid is bordered by middle turbinate and superior turbinate and occasionally by supreme turbinate.

Ethmoid cells are open cranially and are extremely important. These open cells and clefts are effectively closed by extensions of the frontal bone, which is termed as Fovea ethmoidalis. The bony roof of the ethmoid is thus provided primarily by frontal bone, which is of great significance for the behavior of fracture, thus appearance of CSF fistula in iatrogenic injuries. An area that deserves special attention is the ethmoid roof. From its orbital plate, the frontal bone sends an extension across the ethmoids, which are open superiorly, to join with the lateral lamella of the cribriform plate.

The extension of frontal bone forms the ethmoid roof, which is indented by various ethmoid air cells and clefts to form indentations or foveolae: specifically, the foveolae ethmoidales ossis frontalis. The ethmoid roof may vary in its orientation from being nearly horizontal to nearly vertical, however, in most patients, the ethmoid roof lies above the level of the cribriform plate, and therefore, the roof has a superomedial aspect. The medial aspect of the ethmoid roof is formed by the lateral lamellae of the cribriform plate, also known as the lamina lateralis of the lamina cribrosa because it projects superiorly or

superomedially from the cribriform plate. Keros has described three types of skull-base conformations that have clinical relevance in sinus surgery. In type one, the olfactory sulcus is 1 to 3 mm deep, the corresponding lateral lamella is short, and there is a significant portion of frontal bone that backs the ethmoid roof, making the roof thick and the sinus less hazardous to operate in. In type two, the olfactory sulcus is 3 to 7 mm deep, and the corresponding lateral lamella forms a considerable portion of the medial ethmoid roof. In type three, the olfactory sulcus is 7 to 16 mm deep, and the ethmoid roof lies at a significant level above the cribriform plate.

The thin lateral lamella is a much larger component of the roof, and a significant portion of the ethmoid roof is not backed by thick frontal bone, making this the most hazardous sinus to operate in. Extreme caution must be exercised when operating along the skull base, especially medially in the region of the thin lateral lamellae of the cribriform plate.

In anatomic study using microscopic techniques, the extension of frontal bone that backs the ethmoid roof measured 0.5 mm,while the lateral lamella was noted to be only 0.2 mm thick. At the ethmoidal sulcus, a groove in the lateral lamella for the anterior ethmoidal artery, the bone measured only 0.05 mm, a 10-fold reduction in the thickness of the roof. This site has been implicated as the most common site for cerebrospinal fluid (CSF) leak during endoscopic sinus

surgery. The anterior ethmoid artery is an important structure of the anterior ethmoid roof. As the artery enters the ethmoid from the orbit, it courses across the ethmoid roof, either in a bony canal at or just below the level of the roof. Often, however, it can lie 1 to 3 mm below the roof on a mesentery. It courses anteriorly as it crosses from lateral to medial, and penetrates the lateral lamellae to enter the olfactory sulcus. Appreciating the subtleties of anterior ethmoid anatomy can be very helpful in endoscopic sinus surgery. Identifying and avoiding dissection on the vessel can reduce the risk of bleeding and orbital hematoma, decrease the chance of skull base injury with CSF leak, and aid in the identification and dissection of the frontal recess.

FRONTAL RECESS AND SINUS

The frontal sinus drains into the middle meatus and nasal cavity through a complex passage. Review of the anatomic nomenclature of this region has produced much discussion. Several authors describe a “nasofrontal duct” that forms the nasofrontal connection. Anatomic dissection reveals that a true duct, that is, “a tubular structure conducting any fluid ” does not exist. In an attempt to refine the nomenclature and more accurately characterize the anatomy, the term frontal recess has been recommended. The frontal recess is the most anterosuperior aspect of the anterior ethmoid sinus that forms the connection with the frontal

sinus. The boundaries of the frontal recess are the lamina papyracea laterally, the middle turbinate medially, the posterosuperior wall of the agger nasi cell (when present) anteriorly, and the anterior wall of the ethmoid bulla posteriorly. If the anterior wall of the ethmoid bulla does not reach the skull base and form a complete posterior wall, the frontal recess may communicate with the suprabullar recess.

The frontal recess tapers as it approaches the superiorly located internal os of the frontal sinus; above the os, it again widens, as the anterior and posterior tables diverge to their respective positions. An hourglass-like appearance is evident, with the narrowest portion being the frontal ostium. There is tremendous variation with respect to the pattern of the nasofrontal connection.

The anatomic complexity of this region is better understood when the effect of the surrounding ethmoid cells, such as the agger nasi cell, frontal cells, and supraorbital ethmoid cells, are considered. An intimate relationship therefore exists between the agger nasi cell and the frontal recess. Secretions from the frontal sinus destined for the nasal cavity usually follow a path through the frontal recess and over the posterior and medial surface of the agger nasi cell. If the agger nasi cell is extensively pneumatized, the frontal recess can be relatively narrowed, and hence the patient may be predisposed to frontal sinusitis. In surgery, an extensively pneumatized agger nasi can be mistaken for the frontal recess or sinus. If a large

agger nasi cell is opened and mistaken for a frontal sinus, the residual superoposterior wall of the agger nasi cell can scar posteriorly to the ethmoid roof, and iatrogenic stenosis or obstruction of the nasofrontal connection can occur. In addition to the agger nasi cell, there are other ethmoid cells that have an intimate relationship with the frontal recess.

Van Alyea reported that approximately 50% of anatomic specimens had anterior ethmoid cells that encroached into the frontal sinus, and that one-third of these encroached into the area of the frontal ostium. He termed these cells “frontal cells” Schaeffer pointed out that anterior ethmoid cells could pneumatize sufficiently into the frontal sinus to give the appearance of duplication of the sinus. Stammberger points out that “from the frontal recess, anterior ethmoid cells can develop into the frontal bone alongside the frontal sinus.” These were called “the bulla frontalis” by Zucker .

The supraorbital ethmoid cell is another anatomic variation in the region of the frontal recess. Supraorbital ethmoid cells commonly occur from pneumatization of the orbital plate of the frontal bone by ethmoid air cells. Kasper felt that these cells originated in the third and fourth frontal furrow regions, from which they pneumatized laterally and superiorly over the orbit into the orbital plate of the frontal bone. Pneumatization of the orbital plate of the frontal bone can also occur, however, from the frontal sinus proper. In such cases, the embryologic

region that pneumatizes the cortex of the frontal bone between the anterior and posterior tables can also pneumatize the orbital plate of the frontal bone. The ventilation and drainage of the frontal sinus and the pneumatized orbital plate would therefore be through a common ostium at the frontal recess. Kasper has described such pneumatization as originating solely from the second frontal furrow. In such cases, opening the frontal recess would provide drainage to the entire region. Stammberger states that the supraorbital cell is ananatomic variation that develops as an extension from the posterior aspect of the frontal or suprabullar recess.

The anatomic complexity of the frontal recess is further highlighted by Stammberger, who points out that most anterior ethmoid cells can develop from the frontal recess. Accordingly, the agger nasi cell, frontal cells, and concha bullosa have been cited to arise from the frontal recess. Therefore, the very structures some authors use to describe the boundaries and limits of the recess can actually form the recess.

Ascribing strict boundaries to define the frontal recess becomes an absurd exercise in light of the high inter subject variability and the far-reaching communications of this space.

SPHENOID SINUS

Sphenoid Sinus is located centrally within the skull. The sphenoid sinus borders several important structures. Lateral to the sinus lie the carotid artery, the optic nerve, the cavernous sinus, and the third, fourth, fifth, and sixth cranial nerves. If the sphenoid sinus is well pneumatized, the optic nerve and carotid artery can indent the sinus, which is covered only by thin bone. In some cases, these structures are dehiscent. Dissection within the sphenoid sinus has understandably been associated with inadvertent carotid and optic nerve damage.

The left and right sphenoid sinuses are separated by the inter sinus septum. A highly variable structure, it can be obliquely rather than sagittally oriented. Minor, incomplete septations of the sphenoid sinus are also common. Manipulation of any sphenoid septation should be undertaken with the utmost care, as the septations and the intersinus septum have been noted to attach off the midline near or on the bony canal of the carotid artery.

PHYSIOLOGY

Cerebrospinal fluid (CSF) consists of a mixture of water, electrolytes (Na+, K+, Mg2+, Ca2+, Cl-, and HCO3-), glucose (60-80% of blood glucose), amino acids and various proteins (22-38 mg/dL). Cerebrospinal fluid is colorless, clear, and typically devoid of cells such as polymorphonuclear cells and mononuclear cells (<5/mm3).

The primary site of CSF production is the choroid plexus, which is responsible for 50-80% of its daily production. Other sites of production include the ependymal surface layer (up to 30%) and capillary ultrafiltration (up to 20%). Cerebrospinal fluid (CSF) represents the end product of the ultrafiltration of plasma across epithelial cells in the choroid plexus lining the ventricles of the brain. A basal layer Na+/K+ ATPase is responsible for actively transporting Na+ into epithelial cells, after which water follows across this gradient. Carbonic anhydrase catalyzes the formation of bicarbonate inside the epithelial cell. Another Na+/K+ ATPase lining the ventricular side of the epithelium extrudes Na+ into the ventricle, with water following across this ionic gradient. The resulting fluid is termed cerebrospinal fluid (CSF).

Cerebrospinal fluid (CSF) is produced at a rate of approximately 20 mL/hr for a total of approximately 500 mL daily. At any given time, approximately 90-

150 mL of CSF is circulating throughout the CNS. Cerebrospinal fluid (CSF) produced at the choroid plexus typically circulates from the lateral ventricles to the third ventricle via the aqueduct of Sylvius. From the third ventricle, the fluid circulates into the forth ventricle and out into the subarachnoid space via the foramina of Magendie and Luschka. After circulating through the subarachnoid space, CSF is reabsorbed via the arachnoid villi.

Circulation of CSF is maintained by the hydrostatic differences between its rate production and its rate of absorption. Normal CSF pressure is approximately 10-15 mm Hg, and elevated pressure constitutes an intracranial pressure (ICP) greater than 20 mm Hg.

AETIOLOGY

In the adult patient, broadly classifying CSF rhinorrhea into the following two categories is helpful: Spontaneous CSF rhinorrhea and CSF rhinorrhea that is secondary to a suspected or known skull base defect. Cerebrospinal fluid leaks that are secondary in nature fall into the categories of trauma, iatrogenic, and tumor-related.

AETIOLOGICAL CAUSES OF CSF RHINORRHOEA

I. CONGENITAL

Meningocele or meningoencephalocele

Congenital skull base defects

Congenital hydrocephalus

II. ACQUIRED

A. IDIOPATHIC

Causes unknown possible intermittent increase in intracranial pressure

B. TRAUMA

a) Surgical

Intranasal surgery

Endoscopic sinus surgery

Transcranial approaches including surgery of the middle and posterior cranial fossa

b) Non surgical

Skull base fractures

Open or penetrating injuries

Post traumatic hydrocephalus

C. INFLAMMATORY

Erosive lesions

1. Mucoceles
2. Polypoid disease
3. Cystic fibrosis
4. Fungal sinusitis
5. Osteomyelitis

Post infective hydrocephalus

D. NEOPLASM

Neoplasm invading the skull base

Intracranial abnormalities causing hydrocephalus

Also classified as by OMMAYA et al (1968)[16]

I. TRAUMATIC

1. ACCIDENTAL

Acute

delayed

2. IATROGENIC

Acute

delayed

II. NONTRAUMATIC

1. HIGH PRESSURE

Tumours Direct

Indirect

Hydrocephalus

2. NORMAL PRESSURE

Congenital anomalies

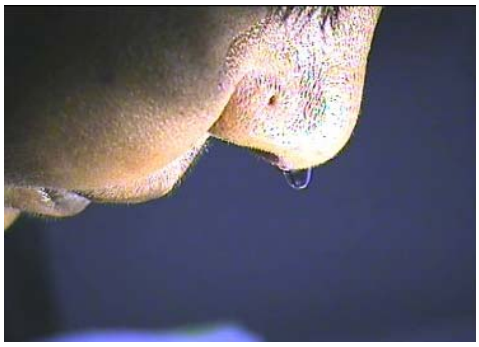
Focal atrophy

Olfactory

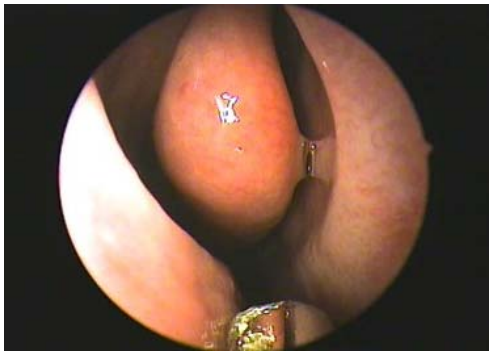
Sellar

Osteomyelitic erosion

Idiopathic

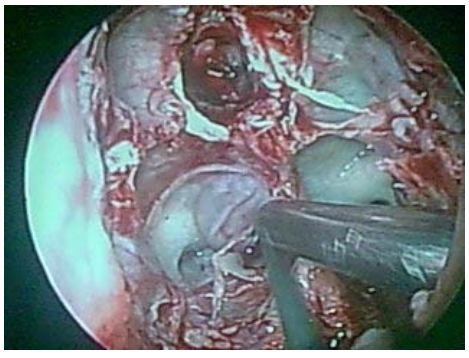


Reservoir sign (figure.3)



Cribriform plate leak (figure. 4)

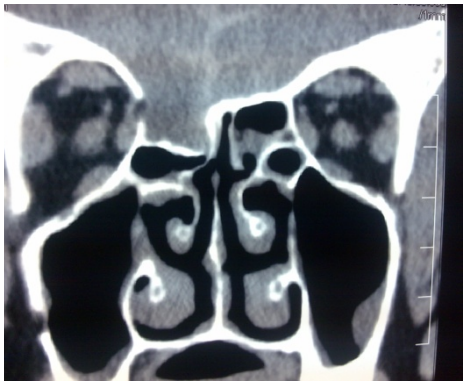
Sphenoid leak (figure. 5)



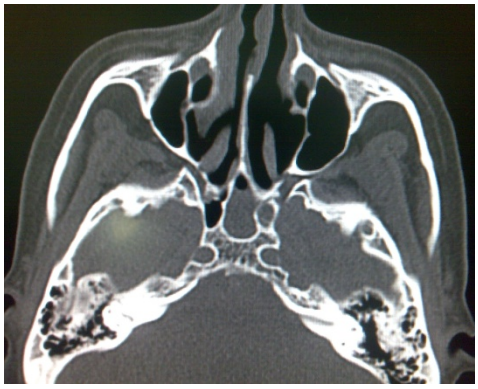
Anterior ethmoid leak (figure.6)

Congenital meningocele (Figure.7)

Traumatic posterior frontal table leak (figure.8)



Traumatic posterior ethmoid leak (figure.9)



Traumatic sphenoid leak (figure.10)

PATHWAYS OF CSF LEAK INTO THE NOSE

Anterior cranial fossa via

1. Frontal sinus
2. Ethmoid sinus/ cribriform plate
3. Sphenoid sinus

Middle cranial fossa via

1. Sphenoid sinus
2. Mastoid air cells/middle ear and Eustachian tube

Posterior cranial fossa via

1. Sphenoid sinus
2. Mastoid air cells/ middle ear and Eustachian tube

Nonsurgical trauma[17]

Penetrating and closed-head trauma cause 90% of all cases of CSF rhinorrhoea. Cerebrospinal fluid rhinorrhoea following a traumatic injury is classified as immediate (within 48 h) or delayed. Of patients with delayed CSF leaks, 95% present within 3 months after the insult. Most patients with CSF leaks secondary to accidental trauma (eg, motor vehicle accidents) present immediately.

In contrast, only 50% of patients with iatrogenic CSF leaks present within the first week.

Surgical trauma[17]

Surgical trauma usually occurs during endoscopic sinus surgery or during neurosurgical procedures. In patients who are undergoing endoscopic sinus surgery, the site of injury is most frequently the lateral cribriform lamella, where the bone of the anterior skull base is thinnest. Other common locations include the posterior fovea ethmoidalis and the posterior aspect of the frontal recess. Skull base injuries vary from simple cracks in the bony architecture to large (>1 cm) defects with disruption of the dura and potentially brain parenchyma. Neurosurgical procedures that result in CSF rhinorrhoea include transsphenoidal hypophysectomy and the endoscopic resection of pituitary and suprasellar masses.

Tumor-related CSF rhinorrhoea

The growth of benign tumors does not commonly result in CSF rhinorrhoea. However, aggressive lesions (such as inverted papilloma) and malignant neoplasms can erode or invade the bone of the anterior cranial fossa. The enzymatic breakdown or destruction of the bony architecture results in

inflammation of the dura and potential violation of the dura by the tumor. If this does not present with CSF rhinorrhoea, very frequently the resection of these tumors results in immediate CSF rhinorrhoea that is typically repaired at the time of the resection, either transcranially or endoscopically.

Congenital

Defects in the closure of the anterior neuropore can result in the herniation of central nervous tissue through anterior cranial fossa skull base defects. Typically, these present through the fonticulus frontalis or the foramen cecum. Meningoencephaloceles typically present in childhood as external nasal masses or intranasal masses seen on examination.

Spontaneous CSF rhinorrhoea[18]

Spontaneous CSF rhinorrhoea occurs in patients without antecedent causes already discussed. This terminology seems to imply that spontaneous CSF leaks are idiopathic in nature; however, recent evidence has led us to realize that spontaneous CSF rhinorrhoea is in reality secondary to an intracranial process, namely elevated intracranial pressure (ICP). The causes of elevated ICP can be multifactorial; nevertheless, once elevated ICP develops, the pressure exerted on areas of the anterior skull base (eg, cribriform, lateral recess of the sphenoid sinus) result in remodeling and thinning of the bone. Ultimately, the bone is weakened

until a defect is formed. At this point, the dura begins to herniate through the defect (meningocele). If a defect is large, brain parenchyma may be herniated as well (encephalocele).

PATHOPHYSIOLOGY[19]

In cases of an immediate leak, a dural tear and a bony defect or fracture has occurred. Possible causes of a delayed traumatic leak are a previously intact dural layer that has slowly become herniated through a bony defect, finally tearing the dura and causing the leak. According to another theory, the tear and bony defect are present from the time of the original injury, but the leak occurs only after the masking hematoma dissolves.

Spontaneous CSF rhinorrhoea usually manifests in adulthood, coinciding with a developmental rise in CSF pressures with maturity. The dura of the anterior cranial base is subject to wide variations in CSF pressure because of several factors, including normal arterial and respiratory fluctuations. Other stresses on the dura include Valsalva like actions during nose blowing. This stress can lead to dural injury in areas of abnormalities of the bony floor.

Increased intracranial pressure is not necessary for non-traumatic CSF leaks to occur. Theories for primary non-traumatic CSF leaks include focal atrophy, rupture

of arachnoid projections that accompany the fibers of the olfactory nerve, and persistence of an embryonic olfactory lumen.

CLINICAL

History[20]

A thorough history is the first step toward accurate diagnosis. The typical history of a CSF leak is that of clear, watery discharge, usually unilateral. Diagnosis is made more easily in patients with recent trauma or surgery than in others. Delayed fistulas are difficult to diagnose and can occur years after the trauma or operation. These cases often lead to a misdiagnosis of allergic and vasomotor rhinitis. On occasion, the patient has a history of headache relieved by drainage of CSF. Drainage may be intermittent as the fluid accumulates in 1 of the para nasal sinuses and drains externally with changes in head position (ie, reservoir sign).

A history of headache and visual disturbances suggests increased intracranial pressure. Sometimes, associated symptoms can assist in localizing the leak. For example, anosmia (present in 60% of individuals with posttraumatic rhinorrhoea), indicates an injury in the olfactory area and anterior fossa, especially when it is unilateral. Interference with function of the optic nerve suggests a lesion in the

region of tuberculum sellae, sphenoid sinus, or posterior ethmoid cells. Patients with recurrent meningitis, especially pneumococcal meningitis, should be evaluated for a defect that exposes the intracranial space to the upper airway regardless of the presence or absence of CSF rhinorrhoea.

Physical examination

Physical examination should include complete rhinologic (including endoscopic), otologic, head and neck, and neurologic evaluations. Endoscopy may reveal pathology, such as an encephalocele or meningocele. Drainage of CSF in some cases may often be elicited on endoscopy by having the patient perform a Valsalva maneuver or by compressing both jugular veins (Queckenstedt-Stookey test). Often physical examination is unrevealing, especially in patients with intermittent CSF rhinorrhoea.

In patients with head trauma, a mixture of blood and CSF may make the diagnosis difficult. CSF separates from blood when it is placed on filter paper, and it produces a clinically detectable sign: the ring sign, double-ring sign, or halo sign. However, the presence of a ring sign is not exclusive to CSF and can lead to false-positive results. In contrast to unilateral rhinorrhoea, bilateral rhinorrhoea gives no clue of the laterality of the defect. However, even in this situation, exceptions can occur. Paradoxical rhinorrhoea occurs when midline structures that act as

separating barriers (eg, crista galli, vomer) are dislocated. This dislocation allows CSF to flow to the opposite side and manifest at the contralateral naris. The clinical findings most frequently associated with CSF rhinorrhoea are meningitis (30%) and pneumocephalus (30%).

INDICATIONS

Unless medical or surgical contraindications exist, surgical repair is recommended in all patients with spontaneous or iatrogenic CSF rhinorrhoea in order to prevent ascending meningitis.

In patients with nonsurgical trauma, a waiting period of 7-10 days to allow conservative measures (bed rest, stool softeners, and lumbar drainage) to assist with spontaneous closure of the traumatic defect is reasonable. However, if CSF rhinorrhoea persists beyond this point, or if a large skull base defect is observed at the time of injury, surgical repair is warranted.

If the operating surgeon has experience with the repair of skull base injuries, a repair should be performed at the time of an iatrogenic surgical injury to prevent long-term infectious complications.

RELEVANT ANATOMY

The most common anatomic sites of cerebrospinal fluid (CSF) leaks are the areas of congenital weakness of the anterior cranial fossa and areas related to the type of surgery performed. According to data from 53 patients with different causes of CSF rhinorrhoea, 39% of leaks occurred in the region of the cribriform plate and air cells of the ethmoid sinus; in 15% of leaks, the fistula extended to the frontal sinus; and in another 15%, the leak was in the area of the sella turcica and sphenoid sinus.

Common sites of injury secondary to endoscopic sinus surgery include the lateral lamella of the cribriform plate and the posterior ethmoid roof near the anterior and medial sphenoid wall. Rarely, the leak can originate in the middle or posterior cranial fossa and can reach the nasal cavity by way of the middle ear and eustachian tube.

CONTRAINDICATIONS

Surgical repair of skull base defects resulting in cerebrospinal fluid (CSF) rhinorrhoea is contraindicated in any patient who is not medically stable to undergo a general anesthetic or comply with postoperative care.

The management of CSF rhinorrhoea depends on the cause, location, and severity of the leak. When trauma is the cause, the interval between trauma and leak is important. The natural history of CSF leak depends on the etiology.

Traumatic leaks often stop spontaneously. The leakage stops within 1 week in 70% of patients, within 3 months in 20-30%, and within 6 months in most patients; leakage rarely recurs. The opposite is true for non traumatic leaks; only one third stop spontaneously, and they tend to persist for several years, with intermittent leakage.

WORK UP

LAB STUDIES

Glucose determination

One of the the main criteria for identification of CSF is based on laboratory quantitative glucose determination. A concentration of 30 mg/ml (1.67 mmol/l) of glucose is considered confirmatory of CSF, if the patient has normal blood glucose levels. However if the the CSF is contaminated by blood then the test is invalid.

A rapid but highly unreliable test is glucose-content determination with the use of glucose oxidase paper. This method of detecting CSF rhinorrhea is not

recommended as a screening or confirming lab test to detect the presence of CSF in the nasal cavity for the following reasons: Reducing substances present in the lacrimal-gland secretions and nasal mucus may cause false-positive results. Glucose, at a concentration of 5 mg/dL, can lead to a positive result with this test. Active meningitis can lower the glucose level in the CSF and may lead to false-negative readings. This test is not specific for the side or site of leak.

Beta-trace protein - also known as prostaglandin D synthase, is synthesized primarily in arachnoid cells, oligodendrocytes, and the choroid plexus within the CNS. Beta-trace protein is also present in the human testes, heart, and serum, making it not specific for CSF. It is altered by the presence of renal failure, multiple sclerosis, cerebral infarction, and certain CNS tumors.

Beta-2 transferrin[21]

Beta2-transferrin is produced by neuraminidase activity within the central nervous system; therefore Beta2- transferrin is located only within the CSF, perilymph, and aqueous humor. The assay has a high sensitivity and specificity, it is performed rapidly, and it is noninvasive. A minimum of 0.5 mL of fluid is necessary for electrophoresis, but difficulties in collection of this fluid have been noted, especially in intermittent, low-volume leaks. Beta-2 transferrin is stable at room temperature for approximately 4 hours; therefore, immediate refrigeration

following collection is recommended as the protein will remain stable for up to 3 days. Specimens should not be frozen. Not specific for side or site of leak; can be difficult to collect if leak is intermittent in nature. This is currently the recommended single lab test for identifying the presence of CSF in sinonasal fluid.

IMAGING STUDIES

Computed tomography (CT) scanning[22]

High-resolution CT scanning is the imaging modality of choice for identifying a skull base defect associated with CSF rhinorrhea.CT scans may demonstrate skull base defects resulting from accidental or iatrogenic trauma, an underlying anatomic or developmental abnormality, or a lesion such as a neoplasm.

CT scans should be performed in the axial plane with 1 mm slice thickness and reformatted into coronal and sagittal planes. The evaluation of congenital defects or spontaneous defects may be aided by 3-dimensional reconstruction of the bone to permit in-depth analysis of the floor of the anterior or middle cranial fossa. Pneumocephalus on a scan suggests a dural tear.

Deviated crista galli is a radiologic sign in patients presenting with primary CSF rhinorrhea; this finding supports a congenital bony dehiscence as the etiologic basis for this condition. In some circumstances, an air-fluid level is present in one

or more of the sinuses. This is not diagnostic of CSF and may be the result of acute or chronic inflammation.

High resolution CT imaging may reveal defects in the skull base that do not leak or are not sites of active leaking, making the diagnosis more difficult.

An axial CT of a patient with a spontaneous CSF leak reveals a defect in the posterior table of the left frontal sinus.

Magnetic resonance imaging (MRI)[23]

Unlike CT imaging, MRI does not delineate well bony defects within the anterior or middle cranial fossa.

Unlike CT imaging, MR imaging is more costly and more time consuming. In many instances, the injection of a contrast agent may be necessary. Like CT imaging, MR imaging may not be localizing.

MRI typically is not recommended as a first line imaging modality in the evaluation of CSF rhinorrhea unless an encephalocele is demonstrated on examination or suspected.

CT cisternography[24]

The diagnostic yield of CT scan can be improved by injecting intrathecal contrast.

The advantages of CT cisternography over conventional CT imaging include more accurate localization and the utility of having to perform only one study. Additionally, the frontal sinus and sphenoid sinus may act as reservoirs for CSF fluid.

CT cisternography depicts the precise location of CSF rhinorrhea in most patients with active leaks.

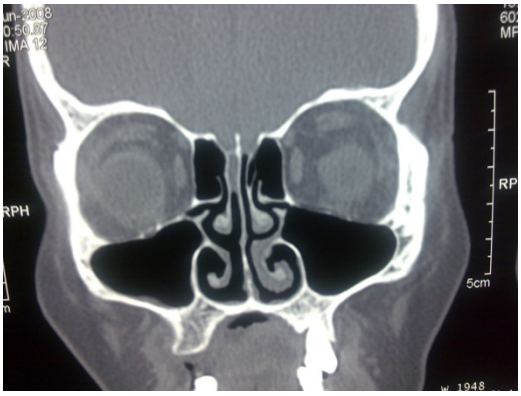
The disadvantages of CT cisternography include the fact that patients with intermittent CSF rhinorrhea may have false negative CT cisternograms and may miss cribriform or ethmoid sinus defects.

This procedure has a low morbidity rate, although nausea, headaches, and acute organic psychosyndromes have been reported. CT cisternography is an invasive procedure.

MR cisternography[25]

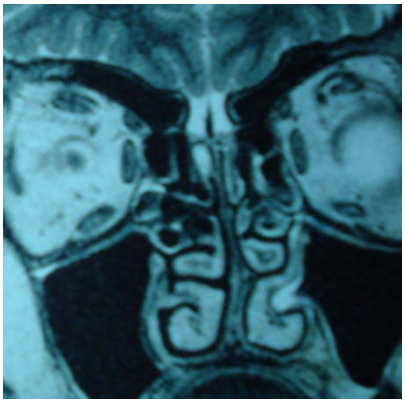
Pulse sequences performed during MR imaging can be designed so as to enhance the probability of detecting CSF within the sinonasal cavity.

T2-weighted imaging can be used to detect the presence of CSF in the sinonasal cavity without the injection of intrathecal contrast.



Cribriform leak in CT without contrast (figure.11)

Cribriform leak with contrast (figure.12)



Cribriform leak in MRI with contrast (figure.13)

Cribriform leak in MRI with contrast(figure.14)

As with CT cisternography, false negative studies may result when CSF rhinorrhea is intermittent.

Nuclear medicine studies

Radioactive isotopes can be introduced into the CSF by means of lumbar or suboccipital puncture. The distribution of these agents then can be determined by using serial scanning or scintiphotography. Another option is to introduce nasal pledgets in various high-risk areas. These pledgets then can be analyzed for the presence of the tracer.

Different tracers, including radioactive iodine-131, radioactive iodinated serum albumin (RISA), ytterbium-169, diethylenetriamine pentaacetic acid (DTPA), indium-111 DTPA, technetium-99m human serum albumin, and 99mTc pertechnetate, can be used. Despite their relative safety, studies based on these tracers have several limitations, as follows: (1) They do not help in precisely identifying the location of the leak. (2) The isotope is absorbed into the circulatory system and can contaminate extracranial tissue. (3) Patient positioning can cause distal pledgets to incorrectly take up the isotope. (4) Readings of radioactivity should be high to determine a true leak; borderline readings are not reliable. False-positive results are present in as many as 33% patients.

DIAGNOSTIC PROCEDURES

The injection of intrathecal fluorescein has been used to diagnose and localize the site(s) of CSF rhinorrhea.

The US Food and Drug Administration has not approved the use of intrathecal fluorescein for the diagnosis or treatment of CSF rhinorrhea.

Precisely 0.1 mL of 10% nonophthalmic solution is diluted in 10 mL of CSF and reinjected into the subarachnoid space over a period of 10 minutes. The use of this dilution and techniques helps to minimize central complications (such as seizures) that have been reported in the past with intrathecal fluorescein.

Nasal endoscopy is performed approximately 30 minutes after an intrathecal injection of fluorescein. In many instances, the surgeon may use this time to perform the initial dissection of the sinuses that would otherwise be performed to gain access to the defect.

In most instances, fluorescein is directly observable within the sinonasal cavity using standard xenon light sources used during endoscopic sinus surgery. However, small defects may leak only a minute amount of fluorescein, and this may be difficult to observe.

The peak absorption range of fluorescein is 494 nm. A blue-light filter (440-490 nm wavelength) can help enhance the visualization of fluorescein, especially in circumstances when fluorescein may be filling an encephalocele or areas of thin bone that could not otherwise be observed with standard xenon light sources.

MANAGEMENT

MEDICAL THERAPY

CONSERVATIVE MANAGEMENT[26]

Conservative treatment has been advocated only in cases of immediate-onset CSF rhinorrhoea following nonsurgical trauma. Conservative management consists of a 7-10 day trial of bed rest with the patient in a head-up position. A head-of-bed position at 15-30° is sufficient to reduce the CSF pressure at the basal cisterns. Coughing, sneezing, nose blowing, and heavy lifting should be avoided as much as possible. Stool softeners should be deployed to decrease the strain and increased ICP associated with bowel movements.

A subarachnoid lumbar drain may be placed to drain approximately 10 mL of CSF per hour. Continuous drainage is recommended over intermittent drainage to avoid spikes in CSF pressure. The long-term consequences of a persistent defect in the anterior cranial fossa dissuade many physicians from deploying this method of treatment (see below).

Antibiotics

The question of the use of prophylactic antibiotics in patients with CSF rhinorrhoea stems from the reasonable assumption that a communication between a

sterile environment (intracranial vault) and a nonsterile environment (sinonasal cavity) will ultimately result in infection of the sterile compartment. However, this assumption has not been easy to study and even harder to prove.

The routine use of prophylactic antibiotics in the case of nonsurgical traumatic CSF rhinorrhoea has been studied in the past with mixed results. A study of 27 patients comparing conservative treatment and transcranial repair revealed that the rates of ascending meningitis in patients treated conservatively were as high as 29%. Nevertheless, that same study showed a 40% rate of meningitis in patients treated via the transcranial route. This was not a statistically significant difference from the rate of 29% in conservatively treated patients. However, 2 recent meta-analyses of patients presenting with nonsurgical traumatic CSF leaks revealed no difference in the rates of ascending meningitis in patients treated with prophylactic antibiotics compared with patients treated with conservative measures alone.

The use of prophylactic antibiotics in patients incurring skull base injuries during endoscopic sinus surgery has not been studied in a randomized controlled fashion. However, administering antibiotics in this setting is reasonable because the skull base injury occurred during surgery for chronic inflammatory/infectious sinusitis and implantation of bacteria into the sterile compartment may have occurred.

Diuretics

Acetazolamide is a nonbacteriocidal sulfonamide that is used primarily as a diuretic. Acetazolamide can be a useful adjunct in the treatment of patients with spontaneous CSF rhinorrhoea associated with elevated intracranial pressure. Acetazolamide inhibits the reversible conversion of water and CO2 to bicarbonate and hydrogen ions.

The relative deficiency of hydrogen ions within epithelial cells results in decreased Na/K ATPase activity, which results in decreased efflux of water into the CSF. Ultimately, this reduces the volume of CSF. The side effects of acetazolamide include weight loss, diarrhea, nausea, metabolic acidosis, polyuria, and paresthesias, any of which may result in the cessation of therapy. When deployed, metabolic profiles should be monitored on a regular basis to ascertain the effect on serum electrolytes.

SURGICAL THERAPY

Surgical options for repair of CSF leaks arising from the anterior skull base can be divided into intracranial and extracranial approaches.

Intracranial repair[27]

Intracranial repair was frequently undertaken (and is still used in select cases) for the routine repair of anterior cranial fossa CSF leaks until the latter part of the 20th century. These leaks typically were approached via a frontal craniotomy. In rare situations, a middle fossa craniotomy or posterior fossa craniotomy was required for leaks arising in those areas. Different repair techniques have been used, including the use of free or pedicled periosteal or dural flaps, muscle plugs, mobilized portions of the falx cerebri, fascia grafts, and flaps in conjunction with fibrin glue. Leaks arising from the sphenoid sinus are difficult to reach by means of an intracranial approach.

Advantages of the intracranial approach include the ability to inspect the adjacent cerebral cortex, the ability to directly visualize the dural defect, and the ability to seal a leak in the presence of increased ICP with a larger graft. When preoperative localization attempts fail to reveal the site of a leak, intracranial approach with blind repair has been successful. In these situations, the cribriform and the sphenoid area, if necessary, are covered with the repair material.

Disadvantages of the intracranial approach include increased morbidity, increased risk of permanent anosmia, trauma related to brain retraction (hematoma,

cognitive dysfunction, seizures, edema, hemorrhage), and prolonged hospital stays. Failure rates for this approach are 40% for the first attempt and 10% overall.

Extracranial repair

Extracranial repair can be divided into external approaches and endoscopic techniques.

External approach

Defects in the posterior table of the frontal sinus may be approached externally via a coronal incision and osteoplastic flap. The osteoplastic flap provides the surgeon with a view of the entire posterior table of the frontal sinus and is especially useful for defects more than 2 cm above the floor and lateral to the lamina papyracea. In select cases, these defects may also be approached with a simpler eyebrow incision and an extended trephination of the frontal sinus in combination with an extended endoscopic frontal sinusotomy. Care must be taken to avoid unnecessary trauma to the surrounding mucosa and the frontal recess entirely.

External approaches to the skull base can also be obtained through various incisions or through nasal approaches for access to the ethmoid sinuses and sphenoid sinus. These include external ethmoidectomy, transethmoidal sphenoidotomy, transseptal sphenoidotomy, and the transantral approach to the

skull base. These procedures are infrequently chosen now given the superiority of the endoscopic approach.

Endoscopic approach[28,29,30,31]

General principles:

Precise anatomical localization of the defect is the most important factor for the success of surgery

Proper preoperative evaluation

Anaesthesia:

General anaesthesia is the preferred mode.

Instruments:

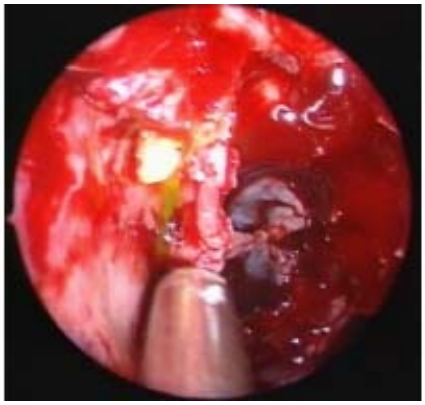
0 and 45 degree Hopkins rod telescope

Light source, camera and monitor

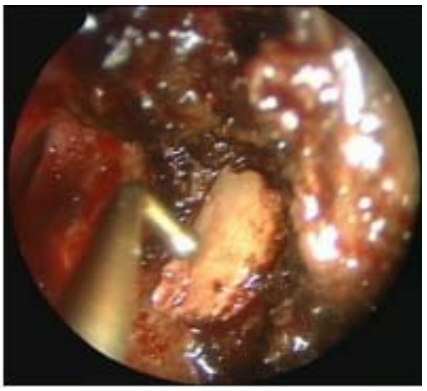
FESS Instruments

Position of the patient:

Supine with 15 degree flexion



Encephalocele in anterior ethmoid (figure.15)



Septal cartilage taken as graft (figure.16)

Fascia lata used as graft (Figure.17)

Final layer with surgicel (figure.18)

Procedure:

Compared with external techniques, endoscopic techniques have several advantages, including better field visualization with enhanced illumination and magnified-angle visualization. Other advantages include the ability to clean the mucosa off the adjacent bone without increasing the size of the defect and accurate positioning of the graft. Multiple studies demonstrate a 90-95% success rate with closure of skull base defects using the endoscopic approach.

The repair begins with the placement of a subarachnoid drain and the administration of fluorescein. Fluorescein is not approved by the FDA for the diagnosis and treatment of CSF leaks. Precisely 0.1 mL of 10% fluorescein is mixed with 10 mL of autologous CSF or bacteriostatic saline and injected through a subarachnoid drain over a period of 10 minutes. The authors have found that injecting this mixture over 10 minutes has resulted in significantly fewer adverse events (eg, seizures) when compared with early reports in the literature.

As previously mentioned, the role of antibiotic prophylaxis has not been studied in a controlled fashion for iatrogenic and spontaneous CSF rhinorrhoea. However, the authors believe that previously published rates of ascending meningitis in untreated CSF leaks is enough to warrant the administration of intravenous antibiotics at the time of the surgical repair.

Decongestion of the nasal cavity with topical oxymetazoline or 4% cocaine solution is recommended in order to maximize endoscopic visualization. Injection of 1% lidocaine with 1:100,000 epinephrine at the root of the middle turbinate and region of the sphenopalatine artery (either transoral or transnasal) helps vasoconstrict blood vessels in these areas and helps to minimize bleeding. The use of intravenous anesthesia with propofol and remifentanil has also been demonstrated to reduce intraoperative blood loss when compared with inhalational anesthesia.

Wide exposure of the defect and any encephalocele is recommended prior to resecting an encephalocele and repairing a skull base injury. This includes performing an adequate maxillary antrostomy, ethmoidectomy, sphenoidotomy, and, if necessary, a frontal sinusotomy. Widely opening the paranasal sinuses can help with visualization and can help prevent iatrogenic sinusitis postoperatively when the nasal cavity is packed with graft material.

For CSF leaks and encephaloceles occurring in the region of the cribriform plate, removing the middle turbinate and sparing this structure for use as grafting material is often helpful. Access to the lateral recess of the sphenoid sinus may require ligation of the sphenopalatine artery, dissection of the vidian nerve, and approach via the pterygomaxillary fossa. Large defects in the sphenoid sinus may require a posterior septectomy for exposure.

A small cribriform plate encephalocele is observed only after removing the middle turbinate.

Once the defect is isolated, the surgeon must ascertain whether an encephalocele is present. If present, the encephalocele is resected using bipolar electrocautery until its stalk can be reduced into the anterior cranial fossa. Resection of the encephalocele is a time-consuming process and must be done in a meticulous manner to ensure that all bleeding is controlled so as to avoid intracranial hemorrhage. Once the encephalocele is resected, the mucosa surrounding the defect must be cleared. This is performed by elevating mucosa away from the defect so as to achieve a margin of 2-5 mm of exposed bone. Bipolar electrocautery should also be performed to help eliminate nests of mucosa that may remain after elevation.

Repair of the defect at this point depends on whether the patient has normal or elevated intracranial pressure and on the size of the defect. Sizing of the defect may be accomplished by placing a flexible ruler next to the defect endoscopically.

A defect in the skull base is measured with a sterile ruler.

Normal CSF pressure[34]

In patients with normal CSF pressure, the size of the defect is less of an issue when it comes to graft selection and method of repair. However, taking into

consideration the size of the defect for the purposes of repair is convenient. The repair of defects less than 2 mm in size is generally successful regardless of the graft chosen.

In this instance, the combination of mucosa (eg, nasal floor, septum, middle turbinate)[32,33] and fibrin glue or fascia (eg, temporalis, tensor fascia lata) and fibrin glue or the use of a fat plug works well. Ensuring that mucosa is not retained within the defect so as to prevent future mucocele formation and that grafts do not obstruct adjacent paranasal sinuses is important. To this end, the authors have recommended widely opening the sinuses prior to skull base repair, and if necessary, the placement of a frontal sinus stent if graft material is used in the frontal recess or adjacent to the frontal recess.

The repair of defects 2-5 mm in size is generally successful with a simple onlay graft of mucosa or fascia. If comminution of the bone around the defect is found, or if a dural injury is significant, the placement of a composite graft is warranted. The middle turbinate bone and mucosa is optimally suited for this type of repair. Bone is placed in an underlay fashion after an epidural pocket is created. Defects greater than 5 mm in size usually require composite grafting or the placement of separate underlay and overlay grafts. The choice of material is similar to that previously mentioned; however, allografts may also be chosen.

Elevated CSF pressure

As with normal pressure defects of more than 5 mm, the repair of encephaloceles and defects resulting from elevated ICP require multilayered grafting. Mastoid tip or septal bone is well suited to serve as an underlay graft. The defect can be reinforced by temporalis fascia or tensor fascia lata and be covered with a free or pedicled mucosa graft.

Septal bone is used as an underlay graft in the repair of this skull base defect in a patient with a spontaneous leak and encephalocele. (Defect measured approximately 7mm.)

Wound closure and postoperative care

Grafts should be anchored to the skull base with the administration of fibrin sealant. The amount of fibrin sealant should be sufficient to anchor the graft yet not obstruct adjacent sinuses or prevent remucosalization of the graft site. The repair is reinforced with gelfoam and nonabsorbable packing to help apply pressure to the graft site.

Preoperative Details

Preoperative CT scans in the coronal plane should be thoroughly reviewed prior to the start of the case. A review of critical anatomy should be performed.

This includes identifying areas of the skull base prone to injury and spontaneous defects: posterior table of the frontal sinus near the frontal recess, the cribriform plate and fovea ethmoidalis, the planum sphenoidale, and, if present, the lateral recess of the sphenoid sinus.

When available, the use of stereotactic image-guided equipment can be calibrated and used intraoperatively to improve navigation and localization during surgery.

Triplanar images help to identify and conceptualize the location of this lateral recess encephalocele.

The surgeon and anesthesiologist should communicate a plan prior to the initiation of surgery so as to avoid untoward events during and after the procedure. A multidisciplinary approach involving otolaryngology, anesthesiology, and neurosurgery is often helpful for the comprehensive care of the patient.

Intraoperative Details

At the end of the surgical case, antiemetics should be administered, and the stomach should be aspirated of blood and fluid to help minimize postoperative nausea and vomiting. The head of bed should be elevated to 15° and the lumbar drain opened to continuously drain 5-10 mL of CSF per hour. If safe, a deep extubation should be attempted and nasal positive pressure is to be avoided.

Postoperative Details

If the repair of the skull base immediately followed an inadvertent injury to the skull base during routine surgery (eg, endoscopic sinus surgery), a head CT scan should be obtained to ascertain the extent of injury to the brain.

Lumbar drainage is performed at 5-10 mL per hour for 48 hours. In patients with known or suspected elevated ICP, the drain is clamped after 48 hours for 6 hours. At this point, an opening pressure is measured. If it is above 20 mm Hg, adjunctive medical therapy is advised (see Diuretics in the Treatment section).

Follow-up

Nonabsorbable packing should be removed 7-10 days after the procedure is performed. Regular endoscopic inspection with minimal debridement of the surgical site should be performed over the long term to identify recurrence of disease.

If the patient is found to have elevated intracranial pressure, the help of a multidisciplinary approach involving an internist, ophthalmologist, and neurologist is invaluable for monitoring patient compliance with adjunctive medication as well as for receiving necessary comprehensive care.

COMPLICATIONS[34]

Knowing the natural course of this condition is important before one examines the results of the various interventions. Meningitis is the most frequent and severe complication of a CSF leak; Streptococcus pneumoniae and Haemophilus influenzae are the most common pathogens. The risk of meningitis during the first 3 weeks after trauma is estimated to be 10%. The rate is 40% in nontraumatic rhinorrhea.

Meningitis caused by a persistent CSF leak is associated with a high mortality rate. Because of the relatively low rate of spontaneous closure, a conservative approach for these indications is not recommended. Spontaneous closure rates vary with the etiology; the recurrence rate after spontaneous closure was 7% in 1 study. The surgical mortality rate is 1-3% for intracranial procedures and is negligible for external procedures. The morbidity for intracranial approaches is clinically significant, with anosmia being the most common complication (10-25% of patients).

MATERIALS AND METHODS

Patients with CSF rhinorrhoea originating from the paranasal sinuses and anterior skull base, from anterior and middle cranial fossa managed in our Upgraded Institute of OtoRhinoLaryngology between 2006 to 2008 were identified and chart reviews performed .

Study design : Prospective Study

Period of study : June 2006 to Nov 2008

All CSF leaks were managed in a multidisciplinary fashion with the involvement of our Institute. As such our review is representative of the institutional experience during the period. This review evaluated the Demographic data, CSF leak site, etiology, management of leaks, surgical complications and recurrence.

Leak site were classified into five locations

-Anterior ethmoid roof

-posterior ethmoid roof

-sphenoid sinus

-cribriform plate

-frontal sinus

CSF leaks were diagnosed and classified accordingly using the following diagnostic methods

1. History
2. Rigid nasal endoscopy[35]
3. CSF analysis
4. High resolution computed tomography(HRCT)
5. Magnetic Resonance imaging
6. Cisternography (CT or MRI)
7. Fluorescein study

Patients of all age group admitted with CSF rhinorrhoea in our institute are taken into the study and investigated with, Glucose estimation in the CSF , Diagnostic Nasal Endoscopy and mandatory CT scan, if doubtful of meningoencephalocele subjected for MRI.

Patients with severe medical illness and who are unfit for surgery were excluded from the study

For the leak in medial lamella, nasal endoscope was introduced between the middle turbinate and septum and for the lateral lamella leak, anterior

ethmoidectomy was done to see the defect and it was sealed without sacrificing middle turbinate. In all cases 0 degree wide angle Hopkins rod telescope was used.

Once the leak was identified, the adjacent area was freshened and fistula repaired using various graft. The repair was further supported by Abgel and Nasal Packing. Nasal pack was removed in the seventh postoperative day.

All patients were put on broad spectrum intravenous antibiotics for a period of one week. Lumbar drain was not given in any case. All patients were on diuretics and anti epileptics for one month. Antitussives and laxatives were recommended in all cases postoperatively. All patients were asked to avoid straining for next three months.

Grafts for repair[36]

1. Free nasal mucosa
2. Pedicled nasal mucosa
3. Bone/ cartilage grafts
4. Temporalis fascia and muscle
5. Adipose tissue

Packing over the graft area was using gelatin sponge and oxidized cellulose. If further stabilization was necessary, non absorbable packing material was used in some cases.

OBSERVATIONS AND RESULTS

The following data is obtained for the present series of 21 patients with CSF rhinorrhea who failed conservative therapy and underwent endoscopic closure at Upgraded Institute of OtoRhinoLaryngology in Government General Hospital attached to Madras Medical college, Chennai during the period of 2006 – 2008.

AGE WISE DISTRIBUTION OF PATIENTS

(Table – 1)

Age	No. of cases
0 to 10	2
11 to 20	4
21 to 30	5
31 to 40	2
41 to 50	5
51 to 60	2
61 to 70	1

SEX WISE DISTRIBUTION OF PATIENTS

(Table – 2)

Sex	No.	Percent
Male	8	36
Female	22	64

AETIOLOGY OF CSF RHINORRHOEA

(Table – 3)

Aetiology	**No.**	**Percent**
Spontaneous	12	54.5
Trauma	9	41
Post meningitis	1	4.5

ASSOCIATION WITH MENINGOENCEPHALOCELE

(Table – 4)

Association	**No.**	**percent**
Spontaneous	5	23
Trauma	4	18
None	13	59

SITE OF LESION

(Table – 5)

Site of lesion	No.	Percent
Cribriform plate	15	68.5
Anterior ethmoid	3	13.5
Posterior ethmoid	1	4.5
Frontal	2	9
Sphenoid	1	4.5

PATIENTS PRESENTATION

(Table – 6)

Presentation	No.	Percent
CSF leak	13	59.5
Cephalocele	1	4.5
CSF leak & cephalocele	8	36

TYPE OF GRAFT USED FOR CLOSURE

(Table – 7)

Graft	No.	percent
Middle turbinate	11	50
Temporalis fascia & middle turbinate	3	14
Fascia lata & middle turbinate	6	27
Septal cartilage & middle turbinate	2	9

CASES THAT NEEDED RECLOSURE

(Table – 8)

	No.
No recurrence	21
Recurrence	1

COMPLICATIONS ENCOUNTERED

(Table – 1)

Complications	**No.**
No complications	20
Recurrence	1
Meningitis	1

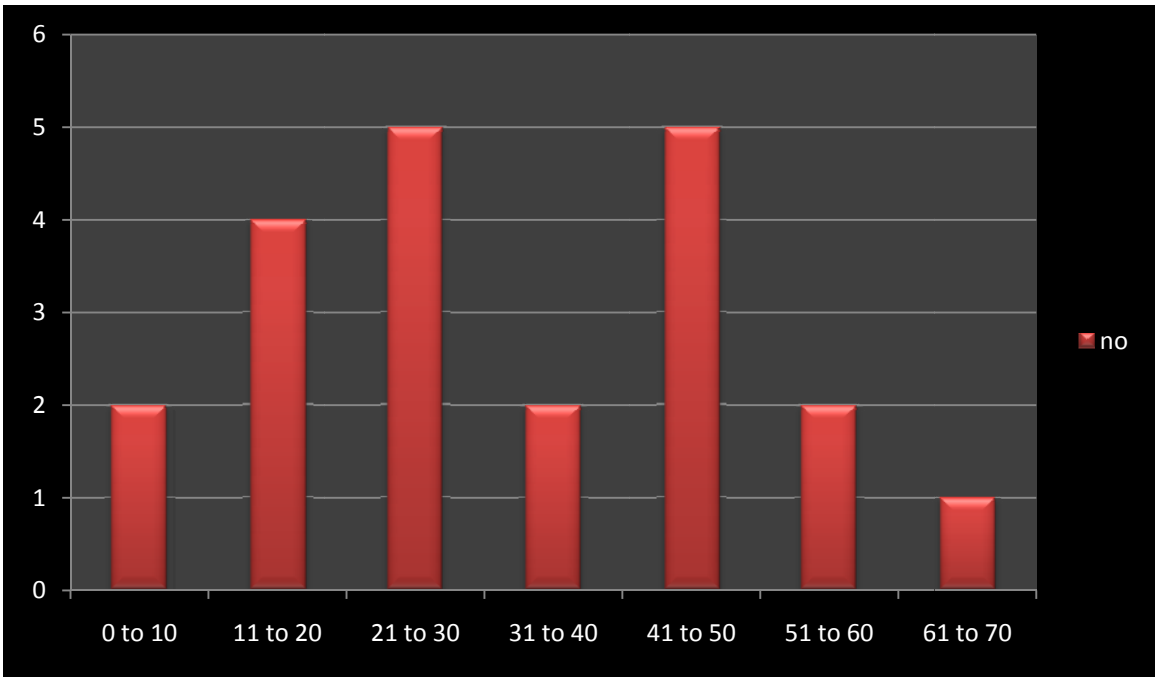


Chart of Table.1, Age wise distribution

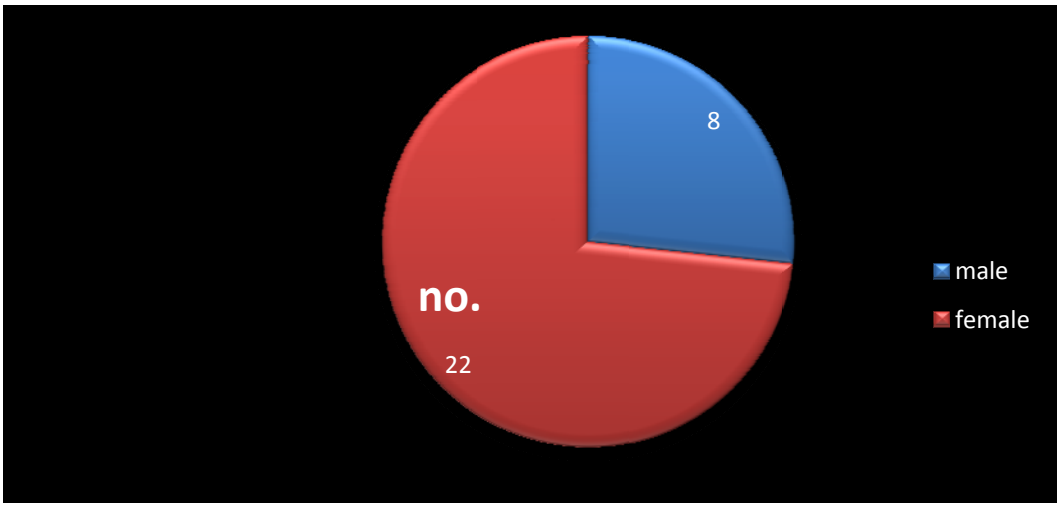


Chart of Table. 2, Sex wise distribution

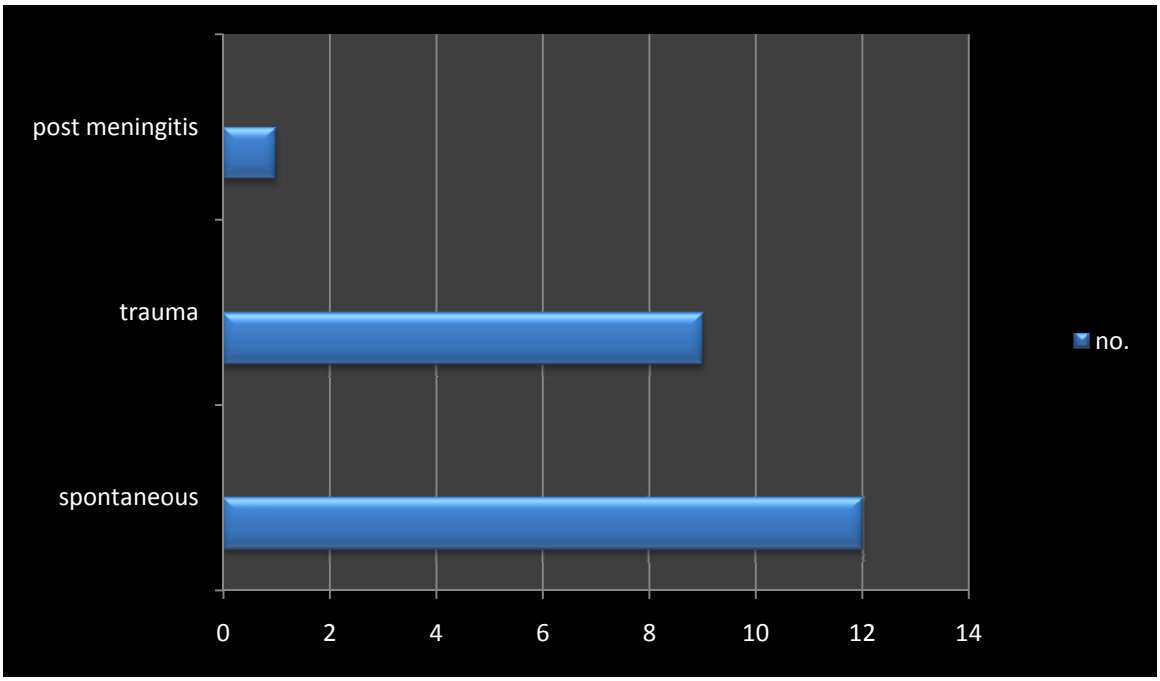


Chart of table 3. Aetiology of CSF rhinorrhoea

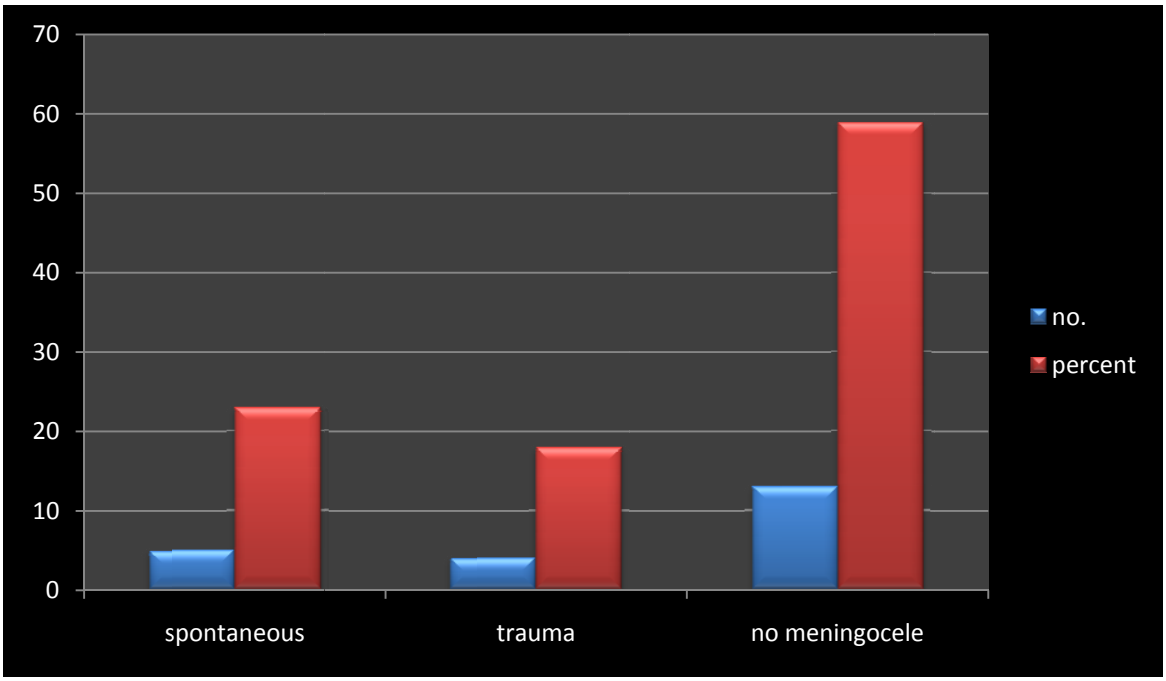


Chart of table 4. Association with meningoencephalocele

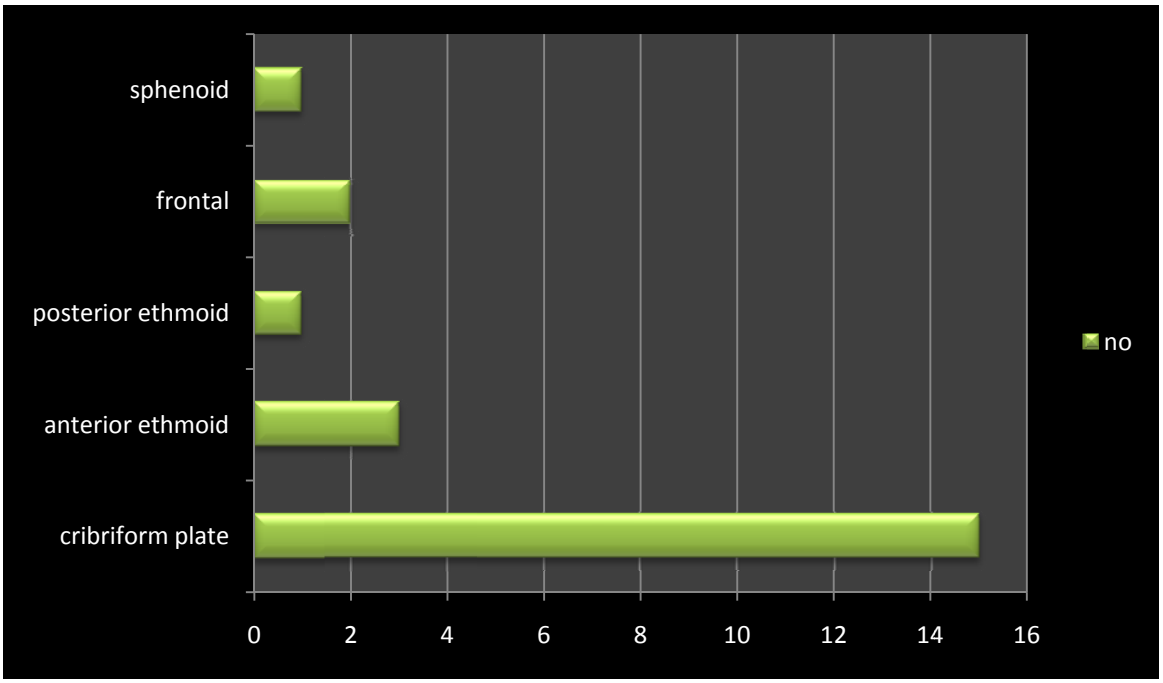


Chart of table 5 - Site of Leak

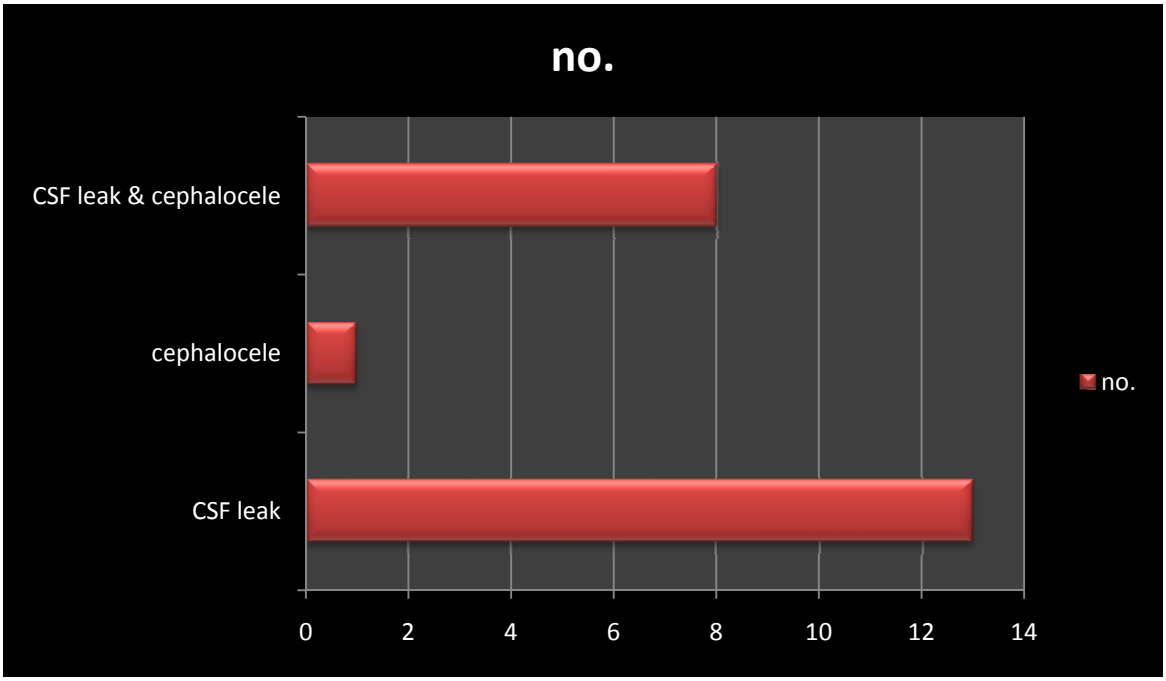


Chart of table 6, Presentation

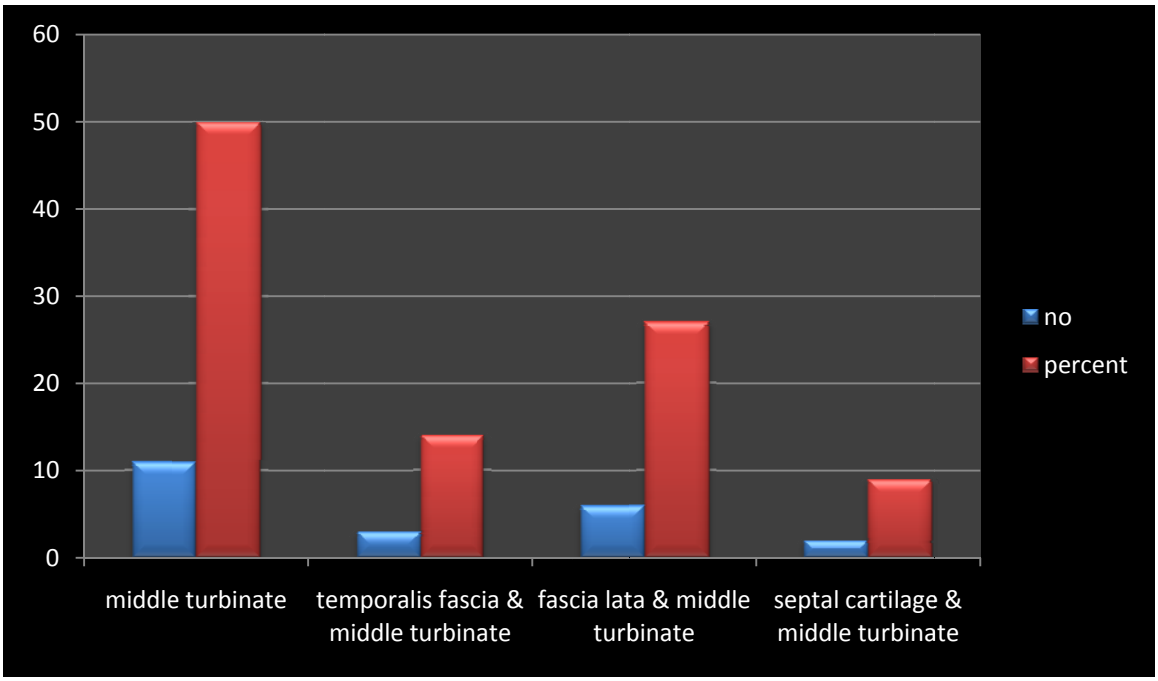


Chart of table 7, Graft used for closure

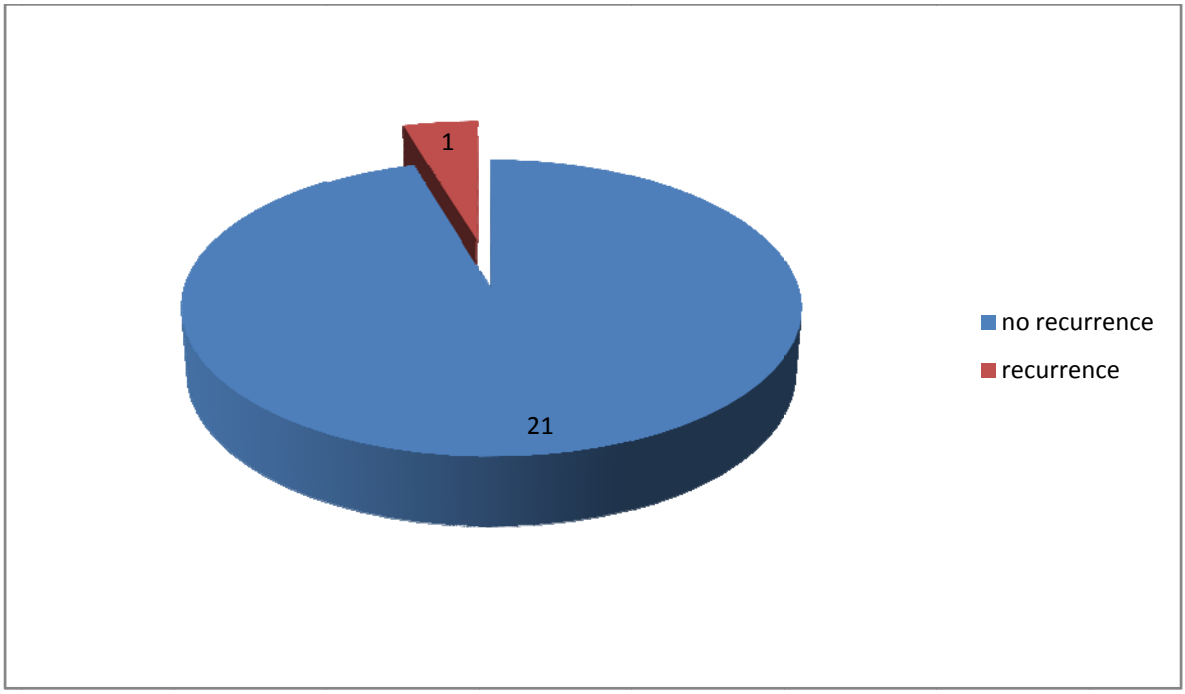


Chart of table 8, cases that needed reclosure

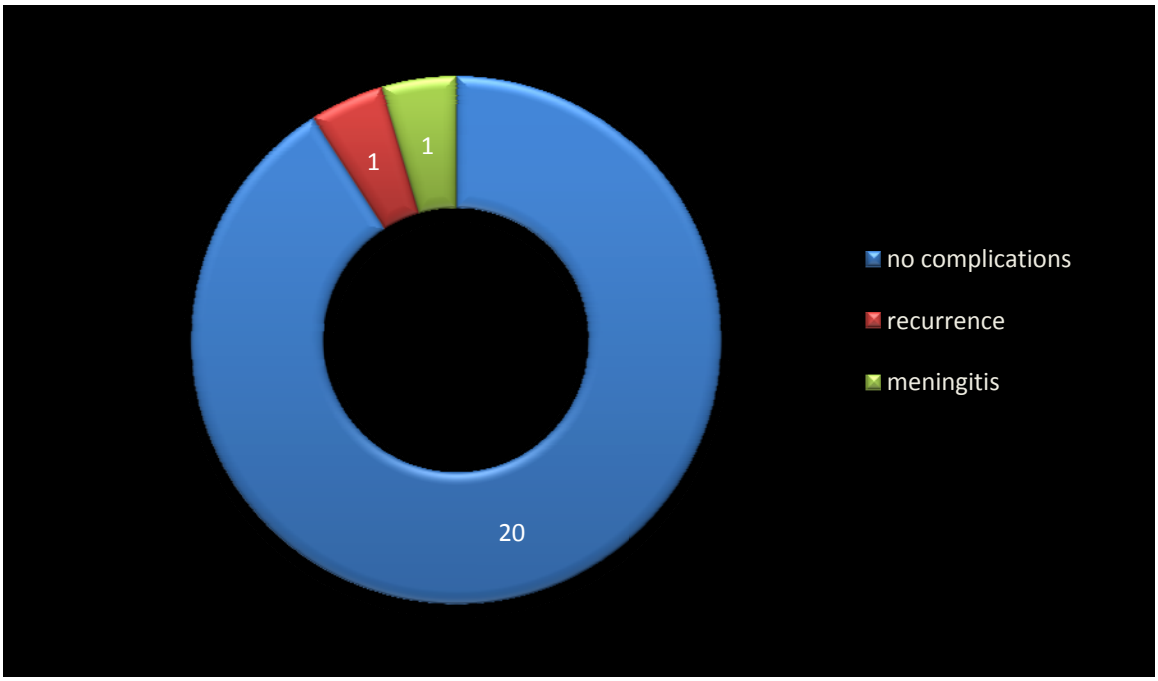


Chart of table 9, complication encountered

DISCUSSION

In our prospective study 22 patients with CSF rhinorrhoea who failed medical and conservative management underwent endoscopic closure of the defect with autologous graft in our Upgraded Institute of OtoRhinoLaryngology, Government General Hospital attached to Madras Medical College.

The socio demographic data and the outcomes of the procedure were analysed and discussed. It is not our intention to compare the results of one approach with another but rather to describe our experience,in the management of CSF rhinorrhoea.

Traumatic and spontaneous cases were preferentially treated with the endoscopic closure. In our case series the frequency of various causes of CSF rhinorrhoea are - spontaneous leaks of 12 patients, while trauma caused 9 leaks and one patient reported after meningitis. Among them 9 patients presented with meningoencephalocele along with CSF rhinorrhoea, while 13 patients presented only with the CSF rhinorrhoea.

OMMAYO et al. (1968)[37] series 80 percent of cases are secondary to head trauma, while 16 percent are the result of operations in the nasal / paranasal

cavities and skull base. Only about 4 percent of cases considered non-traumatic or spontaneous.

D.Richard Lindstrom[38] et al. experience in Wisconsin Medical college is given below in number of patients

Trauma including iatrogenic -42

Spontaneous -13

In our study the demographic data the age group affected lies between 10 to 50 years. Also in our study females are affected more compared to males, causative factor could not be identified.

D.Richard Lindstrom et al. experience in Wisconsin medical college in 53 number of patients, 28 were females and 25 were men. Patient age ranged from 21 to 72 years with mean age at repair of 47.6 years.

In our study the site of defect identified were

Cribriform plate - 68.5%

Anterior ethmoid - 13.5%

Posterior ethmoid - 4.5%

Frontal - 9%

Sphenoid - 4.5%

According to Kennedy[39] site of CSF leaks among 27 patients found to be 17 patients with leak in ethmoids, while 6 patients in the cribriform plate and in the sphenoid in 4 patients.

D.Richard Lindstrom et al. experience in Wisconsin Medical college is given below in number of patients

Cribriform plate – 20

Anterior ethmoid – 10

Posterior ethmoid – 5

Frontal - 6

Sphenoid - 15

In our case series most cases are managed with locally available graft, the middle turbinate(50%). First time repair and small leaks with out meningo encephalocele usually managed with the middle turbinate. Large defect and defect with meningoencephalocele will require multi layered closure, either with septal cartilage, temporalis fascia or fascia lata. The one case that needed revision closure, was closed with the fascia lata.

In our study only one patient needed revision surgery, where the CSF leak recurred after 6 months. All other 21 patients were managed with first attempt closure (95.5 %) .

Dodson et al[40] treated 29 cases of CSF rhinorrhea with endoscopic techniques. Seventy-five percent had resolution after their initial repair. Duration of follow-up ranged from 3 to 43 months. Lanza et al reviewed 36 patients that underwent endoscopic repair of CSF fistulas. During the first attempt, successful endoscopic repair was achieved in 94%.

With Miliand Kirtane experience with 267 patients of endoscopically treated CSF rhinorrhoea , the results of achieving the closure was 96.63% in the first instance and 98.88% after revision surgery.

Most case series have reported success rates of 90 to 95 % with the endoscopic repair of CSF rhinorrhoea. D.Richard Lindstrom et al. experience in Wisconsin Medical college reported success rate of 91% with the endoscopic closure. Also in properly selected patients, non operative management was ultimately successful in 90% of patients. Because all of these therapeutic modalities can be successful, the managing surgeon must consider factors such as leak location, etiology, and closure method prior to repair.

Among 22 patients only one had meningitis in the immediate post operative period, that too managed with antibiotics. Follow up of the patient for about 2 years was normal and without leaks.

Endoscopic closure of CSF rhinorrhoea is strongly recommended. It is safe, simple and with high success rate.

CONCLUSION

The present comprehensive prospective study of CSF rhinorrhoea reviews the outcomes of management and evaluate the different modes of closure which concludes with

- Commonest cause of CSF rhinorrhoea found to be spontaneous in nature, while trauma comes next among our patients.
- 11 to 50 yrs is the commonest age group affected, with about 91 percent of patients falling in this age group. Female are affected more than males in our study.
- Proper clinical history and investigations will identify the leaks and the site of leak and whether there is associated meningoencephalocele or not.
- Spontaneous csf rhinorrhoea found to be higher than traumatic, and most patients either spontaneous or traumatic presented with the meningoencephalocele.
- In our study the most common site of CSF rhinorrhoea found to be in cribriform plate, next being anterior ethmoid while posterior ethmoid, sphenoid and frontal areas relatively less common.

- Our study concludes that successful endoscopic closures of the CSF rhinorrhoea of the presented patients can be achieved with 95.5 % of the patients on first attempt.
- The success reported here is the result of improvement in our understanding of the detailed evaluation preoperatively by imaging methods, endoscopic identification of the leaks exact site and assessment of the defect size and closure with the appropriate graft, either near by the site, or if the size is of bigger one, fascia lata or temporalis fascia will be used and proper postoperative care given.
- In our study only one patients needed the revision closure of the CSF leak.
- Among 22 patients only one patient had meningitis in the immediate postoperative period, which was treated with antibiotics and steroids.
- To conclude, recent advances in nasal endoscopic surgery in the anterior skull base areas have made it the procedure of choice for the repair of CSF rhinorrhoea.

PROFORMA

Case No.

Name : MRD No.

Age : Address:

Sex : .

Occupation : D.O.A ;

Income : D.O.S. ;

Contact No. : D.O.D. ;

PRESENTING COMPLAINTS :

1. Watery Nasal Discharge

2. Headache

3. Meningitis

4. Nasal Mass

5. Fever

6. H/O trauma

7. H/o surgery

8. Other History

Personal history- H/o of allergy to dust and drugs,

H/o asthma,

Family history

Examination

General examination:

Built

Nutrition

Mental status

Pallor/ Icterus/Cyanosis/ Clubbing / Pedal Edema

Lymph Node status

Vital Data

Temperature

Pulse

Respiratory rate

Blood pressure

ENT examination

NOSE

External contour

Mass in nasal cavity

Septal position

Bleeding

Paranasal sinus tenderness

Cold Spatula Test

POST NASAL EXAMINATION :

- Any extension of mass into nasopharynx

EAR Right Left

Pinna

External ear

Tympanic membrane

ORAL CAVITY

Oropharyx

Indirect Laryngoscopic Examination

Eye examination

Face examination

Neck examination

Cranial Nerve Examination

Systemic Examination

Cardiovascular system

Respiratory system

Abdomen

Central nervous system

INVESTIGATIONS

a) Blood – Hb %TC/DC/ESR/BT/CT

Blood Urea/ Sugar / Serum Creatinine

b) Urine – Albumin / Sugar / Microscopy

c) Blood grouping and typing

d) CSF analysis –

Glucose estimation

e) CT PNS

Plane and contrast

Axial and coronal

f) MRI

g) Pre OP Diagnostic Nasal Endoscopy

SURGERY;

Site of defect

Method of closure

Post operative care

FINAL DIAGNOSIS

Post operative follow up

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MASTER CHART

S. no	Name	Age	Sex	Ip. No.	Aetiology	Site of defect	Closure method
1.	Geetha	20	f	6748	Spontaneous, meningo encephalocele	Cribriform plate	Temporalis fascia + MT
2	Faridah	47	f	30841	spontaneous	sphenoid	Fascia lata+ MT
3	Anjalai	19	f	38754	spontaneous	Cribriform plate	Temporalis fascia + MT
4	Elamaran	30	m	10776	spontaneous	Cribriform plate	Fascia lata + MT
5	Nallammal	65	f	17517	trauma	Cribriform plate	Middle turbinate
6	Abibunisha	50	f	22046	spontaneous	Anterior ethmoid	Temporalis fascia + MT
7	Saravanan	26	m	9704	Trauma,meningo encephalocele	Frontal bone	Fascia lata+ MT
8	vinayagam	48	m	64937	Trauma, meningo encephalocele	Cribriform plate	Fascia lata+ MT
9	Janaki	45	f	30829	spontaneous	Cribriform plate	Middle turbinate
10	Balasundaram	28	m	13987	Trauma, iatrogenic	Anterior ethmoid	Middle turbinate
11	Manimaran	22	m	23451	Trauma, meningoencephalocele	Anterior ethmoid	Septal cartilage, MT
12	Nagaraj	18	m	21973	trauma	Cribriform plate	Middle turbinate
13	Valliammal	35	f	20981	trauma	Cribriform plate	Middle turbinate
14	Rabecca	4	fch	55691	Spontaneous, encephalocele	Cribriform plate	Middle turbinate
15	Valarmathy	45	f	55981	Post meningitis	Cribriform plate	Middle turbinate
16	Rajaraman	16	m	77217	Trauma, meningoencephalocel	Cribriform plate	Middle turbinate

					e		
17	Alamelu	33	f	47134	Spontaneous,	Cribriform plate	Middle turbinate
18	Bhuvaneshwari	5	fch	25334	Spontaneous, meningoencephalocele	Cribriform plate	Fascia lata+ MT
19	Sasikala	52	f	22910	Spontaneous, meningoencephalocele	Cribriform plate	Middle turbinate
20	Sridevi	26	f	57656	Spontaneous, encephalocele	Frontal sinus	Fascia lata,MT
21	Sudha	54	f	64529	spontaneous	Cribriform plate	Middle turbinate
22	Kennedy	44	m	78261	Trauma	Posterior ethmoid	Septal cartilage, MT

Nagaraj 18/m 21973 developed Meningitis in the post operative period

Bhuvaneshwari 5/fch 25334 needed revision surgery for recurrence